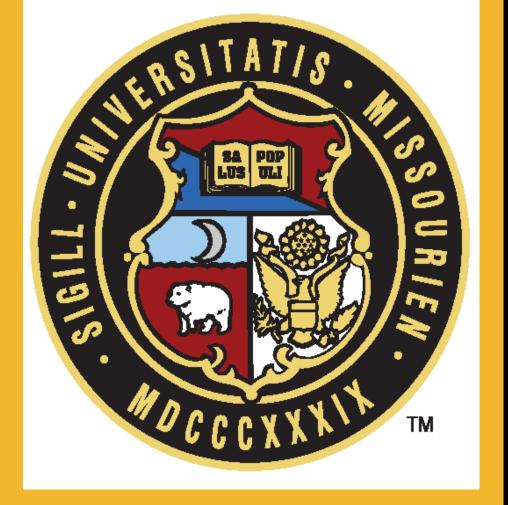


An Unusual Case of Water Intoxication

Amanda Jo Hersh D.O., Keith N. Norton M.D., Chris C. Stacy M.D.

Department of Pathology and Anatomical Sciences, University of Missouri Office of the Chief Medical Examiner of Boone/Callaway County, Columbia, MO 65212, USA



INTRODUCTION

We present the case of a 20-year-old incarcerated male who was discovered dead in his cell. Prior to his death, the decedent was witnessed to drink copious amounts of water, roughly 300 3-4oz cups, over a two-hour time period. During the event, the decedent had one episode of watery, bilious emesis. This was his first incarceration and there was suspicion he had been sexually assaulted by another inmate, which prompted the decedent to "act out" to secure a single cell. Further investigation revealed the man had no significant past medical history, history of head injury, seizure disorders, psychogenic polydipsia, and took no medications, although the possibility of illicit drug use was suspected.

EXAMINATION

At autopsy, pertinent findings included: moderate cerebral edema with mild cerebellar tonsillar herniation, pulmonary edema and congestion, and hemorrhagic pancreatitis.



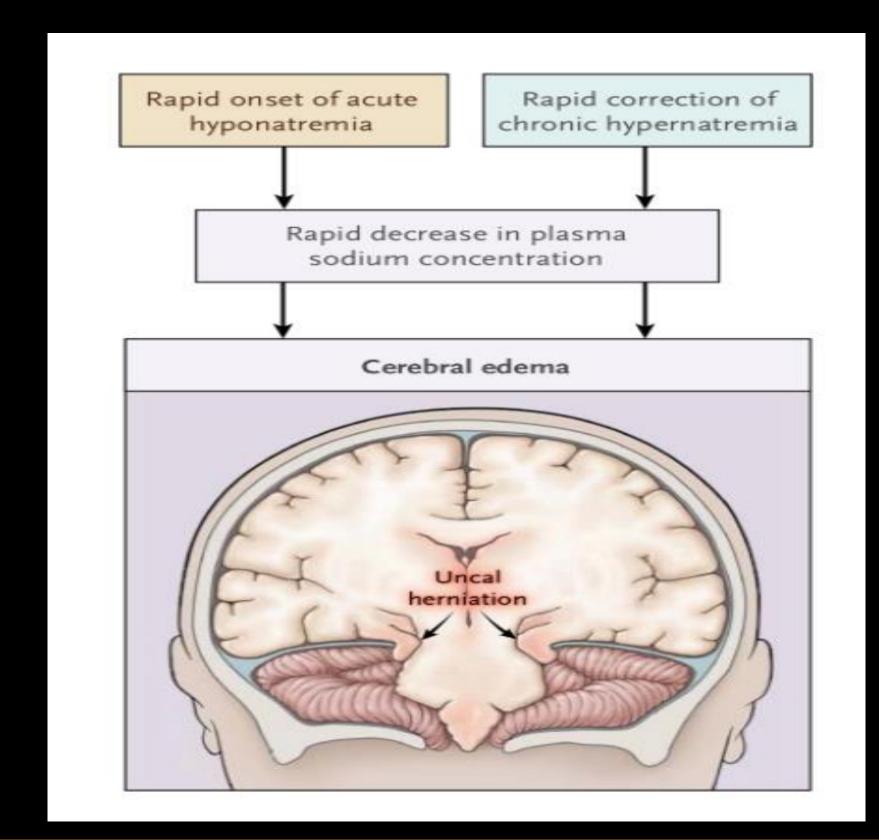
TOXICOLOGY FINDINGS

Vitreous fluid results revealed a sodium of 102 mmol/L (reference range 130-135 mmol/L) and chloride of 101 mmol/L (reference range 105-135 mmol/L). Vitreous urea nitrogen, potassium, and glucose were within normal limits. These values align with the diagnosis of severe hyponatremia (90-105 mmol/L) secondary to acute water intoxication.

compound	<u>Result</u>	<u>Units</u>	Matrix Source
Caffeine	Positive	mcg/mL	001 - IVC (Inferior Vena Cava) Blood
Naloxone	Positive	ng/mL	001 - IVC (Inferior Vena Cava) Blood
Creatinine (Vitreous Fluid)	0.78	mg/dL	003 - Vitreous Fluid
Sodium (Vitreous Fluid)	102	mmol/L	003 - Vitreous Fluid
Potassium (Vitreous Fluid)	14	mmol/L	003 - Vitreous Fluid
Chloride (Vitreous Fluid)	101	mmol/L	003 - Vitreous Fluid
Urea Nitrogen (Vitreous Fluid)	11	mg/dL	003 - Vitreous Fluid

PATHOPHYSIOLOGY OF ACUTE HYPONATREMIA

Acute hyponatremia is hyponatremia that rapidly develops in less than 48 hours. Under normal physiologic circumstances, the brain maintains osmotic equilibrium with plasma osmolality. In this case, the decedent's water intake led to a dilutional hyponatremia which decreased the plasma osmolality. In response to the altered osmotic gradient, extracellular fluid entered the brain causing moderate cerebral edema and impaired cerebral function (hyponatremic encephalopathy). As the cerebral edema increased, the ability of the brain to expand was limited by the skull, leading to cerebellar tonsillar herniation.



<u>Picture Source: Disorders of Plasma Sodium — Causes, Consequences, and Correction</u>. N Engl J Med 2015

MANIFESTATIONS OF HYPONATREMIA

The severity of neurological symptoms reflects the degree of cerebral edema and can be mild with nausea and malaise. The degree of neurologic symptoms correlates to the ability of the brain to control volume regulation. Eventually, as volume regulation diminishes, this can progress to lethargy, obtundation, seizures, coma, and respiratory arrest, especially if sodium concentrations fall below 120 mmol/L.

Hyponatremia: Classification by Neurological Symptoms

	Serum	Neurological Symptoms	Typical Duration of Hyponatremia
severe	<125 mmol/L	vomiting; seizures; obtundation; respiratory distress; coma	acute (<24-48 hrs)
moderate	<130 mmol/L	nausea; confusion; disorientation; altered mental status; unstable gait/falls	intermediate or chronic (>24-48 hrs)
mild	Id <135 headache; irritability; mmol/L difficulty concentrating; altered mood; depression		to many weeks/months)

DISCUSSION